ADAPTATION AND THE CHEMICAL THEORY OF SENSORY RESPONSE

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I. HERING'S THEORY OF THE METABOLISM OF RESPONSE

It is the purpose of the present paper to offer certain amendments to Ewald Hering's theory of the metabolism of response.[1]

This theory, which has been applied by its author principally to the visual and temperature senses, is based upon a consideration of the states of equilibrium and disequilibrium which exist under different conditions between the opposed vital processes of anabolism and katabolism. It is obvious that in a stable organ or organism these processes of constructive and destructive chemical change must approximately balance each other, and when this state of balance is determined by the forces of the living body alone it is called by Hering "autonomous equilibrium."

However, a living body is very seldom unacted upon by outside forces and if such forces cause response this must be because they influence either the constructive or the destructive phases of the metabolism. If a certain constant stimulus increases the velocity of the destructive chemical process it will tend to decrease the amount of destructible chemical substance in the body or organ upon which it is acting. In doing this it will also cause a relative diminution in the amount of such substance which can be destroyed under the influence of the stimulus in a unit of time. In other words, stimulation will diminish the sensitivity of the organ. It follows, moreover, that the rate of the destructive change will decrease constantly until—with the stimulus still acting—it is again exactly balanced by the unstimulated constructive change. This state of affairs is what Hering calls "allonomous equilibrium at low potential."

It is clear that an entirely analogous line of reasoning will follow from the conception of a stimulus whose effect is to augment the constructive rather than the destructive phase of the metabolism. The equilibrium which is finally reached

under the action of such a stimulus is called by Hering "allonomous equilibrium at high potential."

The chemical condition of an organ in allonomous equilibrium differs quantitatively from that of an organ in autonomous equilibrium. If the allonomous equilibrium is at high potential there is an excess, or if it is at low potential there is a deficiency, of destructible substance in the organ, as compared with the amount characteristic of autonomous equilibrium. When a stimulus is withdrawn after a state of allonomous equilibrium has been reached the organ tends naturally to return to the autonomous level, thus reversing the chemical motion which was brought about by the stimulus. All motion of this sort is change from one state of equilibrium to another and is itself allonomous or autonomous according as it is determined by the stimulus or by the internal vital forces, respectively.

Hering's theory of the psycho-physics of response supposes that sensation is conditioned by definite changes in the equilibrium state of the appropriate sense-organ.[2] A stimulus causes sensation by disturbing the equilibrium and thus inviting the metabolic process to seek a new level, but when the latter is found sensation vanishes. When the stimulus is removed the living substance automatically returns to its original state, and during this process, also, there is sensation, usually of a type opposite in quality to that caused by the stimulus. Such shifts in the point of equilibrium always imply alterations in the sensitivity or "potential" of the living substance.

II. The Criticism of Hering's Theory

The theory of the metabolism of response above outlined has some remarkable merits and almost as many remarkable demerits. On the credit side we may count its power to explain the adaptation of sense-organs to specific stimuli, the phenomena of after-images, and the relation of antagonism which exists between different stimuli in certain departments of sensation.

However, Hering's theory has been justly subjected to severe adverse criticism.[3] In the first place, a large part of its explanatory power depends upon the supposition that stimuli encourage anabolic as well as katabolic processes in protoplasm. The arguments which have been brought forward against this supposition are more general than we should wish, but they are nevertheless fairly convincing. Anabolism

is essentially a repair process, a restitution initiated by internal forces to compensate for the destructive effects produced in the organism by stimuli.

There are other more serious objections to Herin's views. One of these is found in the fact that, contrary to the demands of the theory, continued constant stimulation does not invariably end in the disappearance of sensation. Moreover, the present writer has shown experimentally that, at least in the case of vision, although changes in the equilibrium point occur during the process of stimulation, by far the greater part of our seeing must go on under conditions which are approximately those of stable equilibrium. In addition, Hering's theory may be correctly accused of formalism. It takes too little cognizance of the concrete concepts of modern chemistry and physiology, which readily fit into the general form of the theory and at the same time render it more realistic.

III. A Revision of the Chemical Response Theory

It is impossible to seriously question Hering's fundamental postulate, the statement that organic matter is characterized by concomitant destructive and constructive chemical change, and that active organic response must be determined by an augmentation of one or the other of these processes, under the influence of the stimulus. If chemical change occurs it must consist in the synthesis or in the decomposition of some substance, and this substance must have a certain definite *concentration* in the living matter which is being considered. Let us designate this substance by S, and its concentration by s. For convenience we shall confine our attention to the processes which occur in a single living cell, and we shall define s as the absolute quantity of the substance S which is contained in the particular cell which we are to study.

Now since we intend to regard the katabolic phase of metabolism as the only one which is directly affected by the stimuli which act upon the cell we may simplify the general mechanism concerned by supposing that no synthetic changes at all occur within the cell, but that the substance S is supplied to it ready-made, by the blood or lymph. From a formal point of view this *nutrition* of the cell is equivalent to a synthetic process going on within it, since it tends to bring about an increase in the concentration of the substance in the cell. Accordingly, it can be represented by the derivative, ds/dt,

the rate of change of the concentration of S with respect to the time. The katabolic changes within the cell are also representable by a similar derivative, with negative values. For purposes of abbreviation let us write:

(a) $$ds/dt = \dot{s}$$

Now we must suppose that in an undisturbed cell which is metabolically stable the processes of nutrition and katabolism are equal and opposite. Hence, if the rate of nutrition is a, and the rate of katabolism k, we have:

(1) $$\dot{s} = a - k = o;$$

which may be designated as the *equation of rest.* This equation does not mean that the living substance is inactive in the rest condition but merely that its activities are balanced in such a way as to produce no resultant or average change in the concentration of S. The physical chemist expresses practically all states of apparent chemical quiescence as equilibria of this general sort.

It is in accordance with common-sense, as well as with the law of "mass action" in chemistry, to suppose that the rate of "autonomous" decomposition, k, of the substance S will depend upon the amount of S which is present to decompose, that is, it will depend upon the concentration, s. If the relationship in question is a purely chemical one, it will be a proportionality,[4] so that:

(2) $$k = c_k s,$$

where c_k is a constant, the "reaction" which is concerned being of the so-called *irreversible* type and hence being independent of the accumulation of waste products. We shall speak of this expression as *the equation of self-excitation.*

Now, in order to avoid the difficulties encountered by Hering, let us assume that the stimulus, I, always produces a *katabolic change* in the substance S, and has no direct effect either upon the rate of nutrition or upon possible anabolic processes. For simplicity's sake we will make the probable assumption that the increase in the rate of katabolism engendered by the stimulus is proportional to the intensity, i, of the latter, so that if this increase is d, we have:

(3) $$d = c_d i s,$$

which may be called *the equation of response.* d is proportional to s for the same reason that k is so proportional (see equation (2), above).

Thus, when a stimulus is acting, we have, from (2) and (3) and the definition of $\dot{s}$:

(b) $\dot{s} = a - k - d$ or

(c) $\dot{s} = a - c_k s - c_d i s$ or

(4) $\dot{s} = a - (c_k + c_d i)\ s$

This will be known as the *equation of total activity*. The values of $\dot{s}$ which it includes are, of course, in general not the same as those of (1), above.

The term, a, represents the repair, while the term, $(c_k + c_d i)\ s$, represents the destructive process. The first part of the latter stands for what Hering would call autonomous katabolism (or dissimilation), the second part for what he would designate as allonomous katabolism. a is, of course, autonomous.

Now it is obvious that if the second term, above, is larger than the first, $\dot{s}$ will have a negative value, and since $\dot{s}$ is the rate of change of s, this means that s will constantly decrease. But as s decreases the term $(c_k + c_d i)s$ must decrease also, and this change will obviously go on until the last mentioned term becomes equal to a, at which time $\dot{s}$ will become zero, and s will thus cease to vary. In this way there will be established an equilibrium analogous to that represented in equation (1)[5] but involving a stimulus intensity, i. It corresponds, then, with the allonomous equilibrium at low potentid of Hering's theory.[6] Since by definition of an equilibrium state we have: $\dot{s} = 0$, for such an allonomous equilibrium we can write:

(d) $\dot{s} = a - (c_k + c_d i)\ s = o$ or

(5) $$s = \frac{a}{(c_k + c_d i)},$$

the equation of equilibrium sensitivity, which expresses the concentration of the substance, S, in terms of the intensity of the stimulus, I, and certain constants.

It is obvious from the form of equation (5) that the greater the intensity of the stimulus the lower must be the concentration of the substance, S, at equilibrium. Since by equation (3) the degree, d, in which the living substance responds to a stimulus of intensity, i, is proportional to s, s may evidently be appropriately defined as the *sensitivity* of the sense-cell. *Hence we arrive at the general result that the higher the intensity of a stimulus which produces allonomous equilibrium, the lower the point to which the sensitivity of the organ is*

reduced. This result is in accord with the facts of response in many departments of sensation.

Now, in accordance with equation (4) the katabolic component of the process within the sense-cell is always:

$$q = (c_k + c_d i)\ s \tag{6}$$

and since by the definition of an equilibrium state,

$$q = a, \tag{e}$$

it follows that under the assumptions thus far made the rate of the katabolic process at any allonomous equilibrium is independent of the intensity of the stimulus responsible for that equilibrium, and is, accordingly, the same as that at autonomous equilibrium.

The question may now be raised as to whether the repair process, a, actually does remain constant during stimulation. It is in harmony with our general knowledge of vital behavior to suppose that marked destructive changes in the substance of a sensory cell will be accompanied by a *positive increase* in the restitutive process. As a matter of fact, the physiological situation which is involved in the process of nutrition makes it easier to assume that such an increase occurs than to support our previous, mathematically simpler, assumption of a constant repair.

A cell is a portion of space marked off from the lymph in which it is immersed, by a membrane. Through this membrane the sensitizing substance must pass. It is probable that if the molecules of the substance in question can pass into the cell they can also pass out. Since the process under consideration is essentially one of osmotic diffusion the rate at which the substance is penetrating the cell at any instant must depend on its concentration in the lymph, while that at which it is leaving the cell must be proportional to its concentration inside.[7] The net rate of increase in concentration of the substance in the cell will, of course, be the difference between the income and the outgo, and this difference is identical with the a of our equations. Hence, if we suppose the concentration of the substance, S, in the lymph to be a constant we can write:

$$a = r - c_r s, \tag{7}$$

the equation of compensation, where r is the rate of income, and $c_r s$ is the rate of outgo. s, of course, is the concentration of S within the cell. Under the conditions, $c_r s$ must always be less than r.

It is obvious that if this equation is true the rate of nutri-

tion will not be constant, but will increase as the sensitivity, s, of the cell decreases. If we substitute the above value of a in equation (4) we get:

(f) $$\dot{s} = r - (c_r + c_k + c_d i)\ s$$

or, for equilibrium:

(8) $$s = \frac{r}{(c_r + c_k + c_d i)}$$

(The equation of equilibrium sensitivity for compensating response) an equation which has the same general form as (5).

If we now substitute this value for s in equation (6), we have

(9) $$q = \frac{r\ (c_k + c_d i)}{(c_r + c_k + c_d i)}$$

(The equation of equilibrium excitation for compensating response) which states that the rate of the katabolic process at allonomous equilibrium is higher the higher the intensity of the stimulus, i being the only variable on the right-hand side of the equation. From the form of equation (9) it is obvious that q must always be less than r. Equations (7)-(10), inclusive, characterize what may be designated as a *compensating response.*

Expression (9) holds for autonomous as well as for allonomous equilibrium, and for the former since: $i = o$, the expression reads:

(g) $$q = \frac{r\ c_k}{c_r + c_k}$$

The term on the right-hand side of the equation may be described as the *non-compensative response,* since it is natural to say that the compensation is zero when the stimulus is zero. It is thus possible to define the *compensation* itself as the difference between the non-compensative and the actual response under a specified stimulus, i, as follows:

(h) $$u = r\left(\frac{c_k + c_d i}{c_r + c_k + c_d i} - \frac{c_k}{c_r + c_k}\right)$$

When $i = o$, $u = o$; when i is very large $(= \infty)$ $u = r\left(1 - \frac{c_k}{c_r + c_k}\right) = \frac{r\ c_r}{c_r + c_k}$ so that the compensation can vary only between the limits, o and $\frac{r\ c_r}{c_r + c_k}$, the latter term

representing the greatest difference in excitation which can exist between an autonomous and an allonomous response under equilibrium conditions.

Now whether or not a given process of response appears to be compensative must depend upon whether or not the value of u at equilibrium is sufficient to produce a *threshold difference* between the self-excitation and that brought about by the stimulus. In other words, if Δq is the difference limen in question, in order that the response should appear compensating the relation $u > \Delta q$ must hold. If $\left(\frac{r\, c_r}{c_r + c_k}\right) < \Delta q$ the response will always appear to be non-compensating.

When any stimulus whatsoever is applied to a sensory cell in allonomous equilibrium the excitation first rises, and then begins to fall; and it will drop finally to an equilibrium level which is determined by the intensity of the stimulus itself, in accordance with equation (9). The excitation under such conditions of allonomous equilibrium must always be considerably less than that which was produced by the stimulus at the outset. Thus, while a stimulus may at first cause a sensation which differs appreciably from that which is characteristic of the self-excitation of the sensory system concerned, adaptation may so diminish this difference that it becomes, at equilibrium, wholly indistinguishable. When such is the case the response will be to all intents and purposes non-compensating. For a higher intensity of stimulation, however, the equilibrium excitation will be greater, and hence for stimuli of this description the compensation will be apparent. Accordingly, we may argue that in all departments of sense in which the chemical mechanism which we have been considering is active, evidences of the existence of compensating response are not to be expected for relatively weak stimuli. Just how strong a stimulus must be in order that compensation should appear in connection with it must depend upon the magnitude of the difference threshold for the sense under consideration, and upon the nature of the constants in equation (h). It is obvious from the form of the equation in question that for $c_r = o$ the compensation will be zero. The larger c_r the greater will be the compensation for a stimulus of given intensity.

Equation (9) asserts that the excitation at allonomous equilibrium increases as the intensity of the stimulus increases, but the relation which is stated to hold between the two is not a proportionality but an asymptotic function more or less

logarithmic in form. Consequently *equation* (9) *may be regarded as a statement of Weber's law in so far as this law applies under equilibrium conditions.* However, it is the writer's opinion that the Weber relationship—which is known to be only approximately logarithmic—holds not only between the intensity of the stimulus and that of the excitation in the receptor, but for every transition in the sensory-cerebral system.

Let us now consider the somewhat more difficult problem as to the exact process which occurs when a stimulus is applied to an organ whose substance is in autonomous equilibrium. For simplicity's sake we shall confine ourselves to the case of non-compensating response, but the results will be typical of compensating response as well. In accordance with equation (3), when a stimulus, i, is first applied to a sense-cell in autonomous equilibrium it increases the rate of katabolism by an amount: $d = c_d is$, if s is the concentration of the substance, S, under the conditions of this equilibrium. The equilibrium itself is thus destroyed, so that s decreases, and with it d, and a new *allonomous* equilibrium point is reached, the conditions of which have been described above.

The first result which appears, then, is that the effect produced by the stimulus is greatest in the beginning, decreasing relatively rapidly at first, more gradually later on, until it finally reaches a level at which it is constant. In mathematical language, the effect produced by the stimulus decreases *asymptotically* towards an equilibrium condition determined by the intensity of the stimulus.

By use of the calculus it is easy to derive from the above equations the following expression, which shows how the rate of the katabolic process in the stimulated cell varies with the time:[8]

$$q = a + \frac{1}{\mathbf{e}^{(c_k + c_d i)t}} \tag{10}$$

where $\mathbf{e}$ is the base of the natural logarithms, and t is the time which has elapsed after the application of a stimulus of constant intensity, i. Examination of this function shows that as time goes on the value of q approaches a as a limit, but theoretically would only reach it after an infinite period. If we put: $t = \infty$, the term containing it becomes zero, so that: $q = a$, which is obviously the condition for equilibrium. As with all curves of this sort, the rapidity with which the limit

is being approached is less the greater t, and in the region of very large values of t is practically zero.

Expression (10) may be named *the equation of adaptation.*

In a similar manner we can calculate an expression which shows how the autonomous katabolism will increase after the stimulus is removed. The exact form of this expression varies, of course, with the value of s which corresponds with zero time. It is simplest if the value selected is: $s = \frac{a-1}{c_k}$, when it becomes:[9]

$$q = a - \frac{1}{e^{c_k t}}, \tag{11}$$

the equation of recovery. It is obvious that for small values of t the change in the term, $\frac{1}{e^{c_k t}}$ will be relatively rapid, while for large values it will be relatively slow. After the passage of an infinite time the term becomes zero, so that, once more, $q = a$, the condition for equilibrium.

Accordingly, the recovery process, like the process which accompanies stimulation, is asymptotic. The sensitivity of the living substance increases rapidly at first, and then more slowly until autonomous equilibrium is practically reached.

Equations (10) and (11) have been calculated on the assumption that the repair process is constant, as represented in (1)-(7), above. If, on the other hand, equation (7) is introduced into the calculations, expressions will be obtained which differ from (10) and (11), but not in an essential manner.

IV. Interpretation of the Theory

It is obvious that the equations derived above offer an approximate representation and rationalization of the processes of rest, stimulation, fatigue and recovery from fatigue, which occur in various sense-organs. In our subsequent discussion *we shall regard sensation as dependent upon the intensity, q, of the katabolic process at any time.*

Certain of the senses show what may be called *self-activity*, as illustrated, for example, by the familiar " idio-retinal light " in the case of vision. The generic law of such spontaneous excitation is given in equation (2). The apparent absence of this unstimulated activity in some sense departments may be accounted for if we suppose that the value of c_k in these de-

partments is so small that the corresponding sensation falls below the threshold. Another possible explanation for the seeming lack of unstimulated activity may be found in the fact that in many cases an *antagonism* exists between two or more simultaneously active sensory mechanisms, so that the unstimulated activity of one (or more) would wipe out for consciousness the effect of the equivalent unstimulated activities of the others.[10]

The equation of response (3), represents the process of active stimulation while the equation of compensation (7), shows how the rate of repair varies with the sensitivity; (2) and (3) and (7) may be regarded as the fundamental and essential postulates of the theory. It is upon these postulates that the other equations, expressing the conditions of metabolic equilibrium, are directly dependent.

The equation of adaptation (10), shows that when a stimulus of intensity, *i*, is applied to a sensory cell the intensity of the response is not constant, but decreases as the time after the application of the stimulus increases. This decrease, however, itself decreases with the time, so that ultimately a point is reached at which the intensity of the response is practically constant. This condition is that of *equilibrium* with respect to the particular intensity of stimulus which is active. The relation which exists between a given intensity of stimulation, *i*, and the corresponding "equilibrium sensitivity," *s*, of the sensory cell is shown in equation (5), or, for compensating response in (8). It is clear that these equations, which are derived mathematically without further assumptions from (2) and (3), and, for compensating response (7), offer a rationale of the phenomena of sensory adaptation. The equation of recovery (11), on the other hand, which has a similar derivation, represents the process by which the cell returns to the non-adapted state.

The theory of metabolic equilibria above outlined is not presented as an exhaustive analysis of the relations involved in the metabolic process. It is offered simply as a crude generalization of the most fundamental principles underlying these equilibria, and hence can be regarded only as a first approximation to the truth. But, as such it must have a certain value.[11] Crudity of theory is more permissible in a field of measurement, such as psycho-physics, in which mean variations are large than in one where statistical procedure is a less essential part of the methodology.

The nature of further details of the metabolism of response which tend to make the situation more complex than we have

represented it above will be discussed in the appropriate context below.

For convenience in our discussion let us assign names to the variables and constants employed in our mathematical reasoning, as follows: (1) $s =$ the sensitivity, (2) $\dot{s} =$ the resultant activity, (3) $a =$ the repair, (4) $k =$ the self-excitation, (5) $c_k =$ the constant of self-excitation, (6) $d =$ the response, (7) $c_d =$ the constant of response, (8) $c_r =$ the constant of repair, (9) $u =$ the compensation, (10) $i =$ "the stimulus" (stimulus intensity), (11) $q =$ the excitation, and (12) $t =$ the adaptation, or recovery, time.

The characteristic phenomenon of metabolic response is *adaptation. Adaptation may be rigidly defined as a specific but temporary reduction in the sensitivity of a sensory cell under the action of a stimulus of constant intensity.* It is the main thesis of this paper that the phenomenon of adaptation is the most important one which sensory psychology has to consider, and that a careful study of the laws of adaptation in the different departments of sense promises to be the most fruitful means of ascertaining the mechanism operative in these departments. Undoubtedly these mechanisms are not the same for any two senses, but certain general principles must hold for all of those senses which have the same general basis, such as, for example, the chemical. Both the resemblances and the differences between the laws of adaptation in the several sense departments should prove illuminating towards the determination of the exact mechanisms of the senses in question.

V. The Applications of the Theory

Let us now enquire to what extent, if at all, the theory of metabolic response as we have developed it is applicable to the several senses, as we now know them.

We should not rashly assume that all of the senses have the same mechanism, or even that they are all chemical. If adaptation occurs in a particular sense, and if the reduction of sensitivity which this implies depends in the general manner expressed by our equation of adaptation (10) upon the intensity of the stimulus, we shall have reason to suppose that the mechanism of this sense is chemical, and that the theory of metabolic equilibria will apply to it. Such adaptation, however, must bear the ear-marks of a peripheral process, and it must not be permanent in the absence of the stimulus. A sense in which there is no adaptation, or in which what adap-

tation does exist seems to be an adaptation of the attention, or to depend upon physical injury to the sense-organ, probably does not directly involve metabolic processes.

Fatigue may occur in senses which are not chemical, since all vital structures are, so to speak, tender, and when subjected to intensive or continued stress tend to break down, at least temporarily.[12] But the laws of such fatigue should be different from those characteristic of metabolic adaptation. Fatigue of this sort would not be specific with respect to the stimulus and it would probably not show equilibria. Moreover, it would be very much slower in recovery than chemical fatigue.

In how far adaptation of the sort which is typical of metabolic response may be simulated by processes which are not strictly chemical cannot be considered in detail in this paper. The type of adaptation in question, as illustrated, for example, in the case of vision, seems to depend upon the fact that something is being "used up" in the process of excitation. One naturally thinks of this "something" as a substance which is present in the beginning in a definite quantity, thus determining the special sensitivity of the sensory cell, and which is chemically decomposed by the action of the stimulus. Energy might also be stored and reduced in level during stimulation, but it seems hardly reasonable to suppose that the sense-cell needs to supplement the energy of the stimulus with energy of its own. Even if the sensitivity of the cell depends upon the amount of specific substance present within it this substance might conceivably be reduced in concentration asymptotically by some process other than that of chemical decomposition, such as for instance,—in the sense of touch —the migration of molecules or ions through a membrane under the influence of increased pressure. This last mentioned process would follow a law similar to our equation of adaptation.

In all investigation of this sort we are dealing in probabilities. However closely the phenomena which we observe may harmonize with deductions from our hypotheses, it is always at least remotely possible that another hypothesis would explain them as well, if not better.

With this digression let us return to the applications of our theory to the facts of sensory response.

The process of nervous conduction, Hering to the contrary notwithstanding,[13] is probably not a metabolic process. Although the changes which occur during the transmission of

the nervous impulse are no doubt molecular they depend in all probability upon the displacement of ions rather than upon decomposition or synthesis.[14] If these latter processes occur in the course of propagation they are probably immediately reversed. Hence we need not look for adaptation in the conductional aspect of sensory response, but must seek it in the characteristic processes of the sense-organs.

A sense in which adaptation is certainly not a characteristic phenomenon is that of *sound.* Stimulation of the ear with a sound of constant intensity does not result in a decrease in the apparent intensity of the tone or noise, once the *tensor tympani* is adjusted, if the attention is unwavering.[15] This is what we should expect if Helmholtz's resonator theory of audition is correct, since, according to this theory the peripheral mechanism of this sense is purely physical. The theories advanced by Ewald and Meyer,[16] to explain auditory response are also stated in purely mechanical terms, and would not lead us to expect in audition any of the effects which would be characteristic of a chemical sense.

It seems likely that the *labyrinthine senses* are also mechanical, although it is difficult to say whether or not they show adaptation. The experiences of persons who are susceptible to sea-sickness seem, however, to point in the direction of their non-adaptiveness. Adaptation to the motions producing seasickness often occurs, but the length of time required for it to appear shows that it is not sensory.

If we turn to sensations derived from the skin, we find that although there are ample evidences of adaptation in the case of touch and temperature, the *sense of pain* appears to be non-adaptive.[17] Hence we should conclude that the last-mentioned sense is not chemical in its mechanism, and this conclusion is in harmony with the reasonable supposition that pain is due to the direct stimulation of the free nerve endings in the skin (and elsewhere).[18] According to this view, pain has no characteristic peripheral mechanism, and its non-adaptiveness is one with that of pure nervous tissue.

The *sense of touch* or pressure, on the other hand, is remarkably adaptive.[19] So far as extant experimental evidence goes to show, adaptation of touch follows the general laws contained in our theory of metabolic equilibria. That spontaneous excitation exists in the case of touch is probable, and is the basis of Fechner's theory of tactual space.[20] From the point of view of perception, however, the corresponding sen-

sations *mean* the absence of pressure or of an outside stimulus. If for relatively weak stimuli the response of all touch cells finally reduces approximately to the level of the self-excitation, k, (equation (1)), we have an explanation for the fact that diverse pressure stimuli which are constantly effective ultimately become indistinguishable from one another, and from the absence of a stimulus. This principle seems to apply to pressures which do not exceed a certain maximum, such as the contact of the clothes with the body. For higher intensities of stimulation the sensation seems to remain above the neutral level for a long time, as we should expect from our theoretical discussion of compensation, above.

The fact that self-excitation exists in the sense of touch is indicated by the experience which we have upon the removal of a pressure stimulus to which we have become completely adapted, or for which the touch cells are in equilibrium. Since in accordance with the equation of equilibrium sensitivity (5), such an equilibrium means a reduction in the sensitivity of the cells in question the removal of the stimulus must be followed by a marked decline in the intensity of the sensation yielded by these cells, for equation (2) makes the self-excitation of the cells depend upon their sensitivity at the time. This fall of the local response below the normal autonomous equilibrium response provides the basis for our perception that "something is lacking." [21] The disappearance of this feeling as time goes on is accounted for in the equation of recovery (11), which demands the return of the sensitivity of such an adapted region to the normal.

The *muscular and articular senses* seem to be very closely similar, in their mechanism, to the sense of touch. Presumably, like the latter, they are chemical in nature, but nothing definite is known concerning their adaptation processes.

As we have already stated, Hering's theory of response metabolism has been specifically applied by its author to the *sense of temperature*.[22] According to Hering's view, heat favors katabolism in the temperature end-organs, while cold favors anabolism, the corresponding sensations being concomitant with an alteration of the equilibrium point from higher to lower, and from lower to higher, respectively. All equilibrium conditions yield the absence of temperature sensation.

It is clear that this scheme accounts very nicely for the balance which exists between the sensations of heat and cold, and especially for the peculiar facts of temperature adaptation. Everyone is familiar with the fact that a decrease in the sen-

sitivity of the skin to heat means a corresponding increase in its sensitivity to cold, and *vice versa*. However, Hering's theory not only violates the canons of general physiology by joining a definite sensation (that of cold) with an anabolic process,[23] but it also explains the relations of the two temperature responses in terms which, although simple, are inconsistent with the demonstrated separate localization of " heat and cold spots."

If, then, we suppose that response for heat and cold occurs in different end cells, and that, moreover, they are both dependent upon the augmentation of certain katabolic processes, we have to account for the obvious antagonism which exists between them by means of an additional assumption. Heat and cold appear to have been correctly described by Hering as *antagonistic sensations*. As we shall see shortly, they are not the only sensations which belong to this class.

The nerve dissection experiment of Head and Rivers[24] has shown that there are at least two independent systems of temperature sensibility. The system comprised by the heat and cold " spots " gives no evidence of adaptiveness, and the reason for this is easy to see, in as much as the end-organs of this system are insensitive to temperatures lying between 26°C and 37°C. Since the curves of sensitivity of the heat and cold spots do not intersect, the temperature system which they make up can have no " physiological zero," and it is the shifting in position of such a physiological zero which constitutes ordinary temperature adaptation. The limits of sensitivity in this system are probably set by the precise nature of the chemical processes which characterize the end-organs. According to our view the heat and cold sensations yielded by the " spots " are antagonistic, like those of the " epicritic " system, but since only the latter show a physiological zero, it is only in the epicritic system that temperature adaptation can produce a qualitative shift in the sensibility.

Several of the experiments performed by Head and Rivers show that simultaneous stimulation of adjoining heat and cold spots result, under the appropriate conditions, in the inhibition of the heat sensations by the cold, or vice versa.[25] As the antagonism between these two sensations obviously cannot exist in the end-organs, as demanded by Hering's theory, it is necessary to assume that it has a central, or at least a *sub-receptoral* location. Let us assume, then, that the arrangement of the temperature nerves is such as to balance " heat " impulses against " cold," so that the intensity of one impulse

is always subtracted from that of the other before any effect is produced in consciousness.[26]

Thus, if q_h is the excitation of the heat sense at any given time, and q_c is the simultaneous excitation of the cold sense, in a specified region of the skin, the effect upon consciousness will depend upon the arithmetic magnitude of their difference:

(12) $$q_t = q_h - q_c,$$

the quality of the sensation being "heat" if the sign of q_t is positive, and "cold" if it is negative. If this hypothesis as to the relationship which exists between the heat and cold excitations be added to those of our theory of metabolic response it will be seen that this theory offers a satisfactory explanation for the phenomena of temperature adaptation so far as they are known at present.

The nature of temperature response is such that a stimulus is never absent. There is no physical zero for cold and the physical zero for heat never occurs in the environment of organisms. However, for purposes of exposition, we may define a "state of rest" of the temperature system in which

(g) $$q_t = q_h - q_c = o.$$

If the temperature is now increased, q_h becomes larger and q_c smaller, in accordance with the equation of total activity (4), and hence a sensation of heat will be experienced. But the alteration which has occurred in the nature of the stimulus disturbs the equilibrium of both the heat and cold sensory-cells so that the excitation of the former gradually sinks in accordance with the equation of adaptation (10), while that of the latter gradually rises, as demanded by the equation of recovery (11). When equilibrium is re-established in both cells the equation: $q_h = q_c$, must again hold, so that $q_t = 0$. Although the temperature still remains at the higher point the sense of heat has vanished.[27]

In an entirely analogous manner it can be shown that if the temperature is lowered from this, or any other point for which equilibrium has been reached, a sensation of cold should be experienced until equilibrium again sets in, at which time the sensation will disappear.[28]

These deductions from the theory are obviously in general harmony with the facts of temperature adaptation. The deductions in question are based upon the assumption that the "repair," a, is a constant. However the fact that there is a limit (about 11°C.) below which complete adaptation to cold does not occur, and another (about 39°C.) above which complete adaptation to heat is reported to be unattainable, seems

to show that, as in the case of touch the response is really "compensating," but that for the lower intensities of stimulation the compensation falls below the threshold. However, the experimental evidence is still too vague to permit surety upon this point.[29]

The sense of *smell* has long been regarded as a characteristically chemical sense, and there is no sense in which adaptation is more in evidence. There seems to be no moderately intense stimulus to which the olfactory cells will not become completely adapted in a relatively short time.

The fact that there is no manifest self-excitation in the case of olfaction seems at first sight to argue against the view that this sense is subject to our theory of the metabolism of response. However, the objection is not a fatal one, for two reasons: first, because there is evidence that antagonism exists between various phases of olfactory response, so that one species of self-excitation may blot out another; and, *second,* because the equilibrium excitation may lie below the threshold. If this latter supposition were made, an explanation would immediately be provided for the completeness of adaptation to all moderately strong stimuli, since for such stimuli the compensation may be insufficient to raise the equilibrium point above the absolute threshold. According to Nagel[30] complete adaptation does not occur in the case of very strong stimuli, a fact which is again corroborative of our theory of compensative response which demands that for higher intensities the excitations should never fall to a neutral level. Other aspects of olfactory adaptation are in harmony with our theory.

However, the writer holds the opinion that the mechanism of olfaction is a very special one and, although chemical, depends upon conditions which differ from those of metabolic equilibrium. He hopes to discuss this view in a later paper. But it is worth while to note that if olfactory response should ultimately turn out to be made up of component nervous activities,—as in the case of temperature sensation—we should probably have to deal with the relation of antagonism represented in equation (12) above, expressed, of course, in olfactory terms.

In the sense of *taste* adaptation is a well proven phenomenon In this sense, also, we find the relationship of antagonism between the separate qualities which are involved, although the antagonism is perhaps less well defined than in the temperature sense.[31] The four component sensations of "sweet," "sour,"

" bitter," and " salt " appear to be balanced against each other in such a way that the first is to a greater or less degree antagonistic to each of the others, the distinctness of the antagonism to " sweet " increasing in the order in which the latter are named.

It has appeared in the study of the temperature sense that adaptation to cold increases the sensitivity of the temperature system to heat, and *vice versa*. Similarly, it has been found by experiment that adaptation to salt raises the threshold for sweet. Not only this, but it appears that strong stimulation with salt may be followed by a sensation of sweet even in the absence of an adequate stimulus to such sensation. This latter is a negative after-image effect, and is immediately explained in terms of the theory of antagonistic sensations which we have outlined in connection with the sense of temperature and the possible application of which to olfaction we have just considered.

If self-excitations of the " sweet " and " salt " mechanisms exist these self-excitations must be equal and opposite in the state of rest, since in this state neither of the corresponding sensations is present although if it were not for the antagonism both sensations would be above the threshold. Adaptation to salt means a reduction of the sensitivity of the " salt " mechanism in accordance with the equation of equilibrium sensitivity (5). Consequently when the stimulus is removed the self-excitation of this mechanism must fall below that for the " sweet " mechanism, as demanded by the equations of self-excitation and of antagonism. This leaves a certain portion of the self-excited activity of the " sweet " mechanism unbalanced, the residue which is responsible for the negative after-image.[33]

Gustatory adaptation occurs slowly and for strong stimuli is never complete. Accordingly, gustatory response, if actually chemical in mechanism, must be to a high degree compensating.

The most careful of all studies of the phenomena of sensory adaptation have, of course, been made in the field of *visual* psycho-physiology, Hering, himself, having worked largely in this field. However, the phenomena of adaptation in vision do not appear to be such as completely to substantiate Hering's hypotheses concerning the exact mechanism of visual response.[34] These hypotheses suppose the visual system to contain three specific substances which in the state of rest have characteristic metabolic equilibria. Decrease in the concentrations of these three substances is accompanied by sensations

of red, yellow, and white, respectively, while increase is paralleled by the opposite sensations, green, blue, and black, respectively. Now the facts of "color mixture" undoubtedly demand an antagonism between the excitations underlying red and green, and between those underlying yellow and blue, but observation does not support the view that there is antagonism between black and white. For these last mentioned qualities, on the contrary, there is fusion. An attempt has been made by G. E. Müller to remedy this weakness in the theory, but however successful, the attempt can hardly save the doctrine from the general objections outlined in the second section of this paper.[35]

To avoid these objections the present writer has proposed certain alternative hypotheses which form the basis of a theory similar in general structure to that of Hering but, he believes, somewhat less formalistic.[36] According to the author's view, the retinal cones contain four separate substances which are capable of being broken down by the action of light. The rates of decomposition of these four substances are the variables which determine the degree in which the corresponding sensation resembles red, yellow, green, and blue, respectively. However, a sub-receptoral mechanism of antagonism is further postulated, in accordance with which equivalent excitations of the red and green substances eradicate each other and are replaced in the same proportion by a new excitation giving the quality, white. The same relationship is supposed to hold for the yellow and blue excitations.

Now if we suppose that in vision, as in the other senses, self-excitation of the component responses exists, we are forced to assume that those for red and green are equal and opposite, and that those for yellow and blue are in a similar relationship. Under this supposition the so-called idio-retinal light can be referred to the white excitation which results from these two sets of balanced excitations. Such exact balance holds for autonomous equilibrium or for the normal rest condition of the retina.

If the eye is stimulated by a light which increases one of two antagonistic excitations more than it does the other the result will be a sensation corresponding in quality to the most intensive component of the response. As time goes on the intensity of this quality—say red—should fall in accordance with the equation of adaptation, until if the response were non-compensating the excitation corresponding to it would finally again equal that of its less stimulated antagonist—the "green" substance. When this point had been reached the

sensation would be one of white or gray. If the stimulus were now removed the reduction in sensitivity of the "red" mechanism as compared with the "green," as demanded by the equation of equilibrium sensitivity, would cause its self-excitation to sink below that of the "green" substance, so that the quality, green, should predominate in the sensation which forms the negative after-image. Other things being equal, this sensation should be darker than the idio-retinal light, since the total activity is less than that which underlies that light.

The experimental study of visual adaptation proves it to follow the general equations of adaptation and recovery.[37] However, when we come to consider equilibrium conditions we find evidence that, at least for stimuli of fairly high intensity, adaptation does not proceed to a point at which the resultant sensation reduces to neutral gray. Consequently we are forced to assume that in the case of vision, as in most other senses, the response is compensating. Accordingly, the equations for allonomous equilibrium will resemble (8) and (9).

The curves of adaptation and recovery of vision have been studied both for the rods and for the cones of the retina, and have the general form demanded by our equations (10) and (11).[38]

In addition to the various sensations which we have considered above there is another psycho-physical process—or phase of activity—which shows in a most striking way the phenomenon of adaptation.[39] It may seem rather daring to assert that *pleasantness* and *unpleasantness* have a direct chemical basis, but that such is the case is certainly suggested by the existence of affective adaptation. The author has for some time been interested in the view that these fundamental and contrasted aspects of the psychical life—pleasure and displeasure—represent synthesis and analysis, respectively, in the chemical substance of specific cortical centers. He hopes to develop some of the implications of this not entirely novel view in another paper.

However, it is pertinent to note here that if this chemical theory of the physical basis of affection is correct the situation which it represents is not only one in which the fundamental equations of our general theory of chemical response should hold good, but is also one which very closely approximates that contemplated by the original katabolism-anabolism view of Hering. Hence, besides explaining the disappearance

of the affective tone of a perception or sensation by means of our equations of adaptation, we can account for the reversal of the tone which generally follows the removal of the conditioning stimulus, in terms of the "assimilation or dissimilation material" which the physico-affective process has piled up. Pleasant experiences mean the accumulation of synthetized substance in the cortical cells. If the external causes acting upon these cells are taken away this overbalancing mass of anabolic material will favor katabolism, and hence the well-known "affective reaction." In a similar way we can account for the pleasure of "relief" which follows the removal of a painful stimulus or stimulus complex. This is the Hering doctrine, pure and simple.

Affective adaptation is typical and complete only in certain forms of experience, namely those in which the affective tone seems to depend upon novelty. Where the affection is determined by an instinct or some other basic mental "complex" the pleasantness or unpleasantness may be long continued and almost non-adaptive. We must suppose that in such cases there is an external repair process which (as represented in equation (7)) tends to maintain the typical metabolic function of the cortical cell by continually supplying it with the requisite chemical substances, whether synthetic or analytic. The beauty of meaningless music or the unpleasantness of some novel odor, are examples of the affective process at its simplest. With repetition this beauty, or disagreeableness, quickly disappears through adaptation. On the other hand, the pleasure of the erotic consciousness, and the unpleasantness of organic pain are affective processes which are undoubtedly supported by massive and complex psycho-physical mechanisms, of the nature of repair. This latter type of affective response, then, is compensative, like the majority of the sensations. On the whole affection tends to be compensative for low intensities and sensation for high intensities.

VI. Summary and Conclusion

If it is true that the characteristic activities of organic matter are at bottom chemical it is important for the psycho-physiologist to investigate theoretically the relationships which exist between the processes of metabolism and those of sensation. Hering has done this with great generality and with significant results, but the development of physiological psychology since his views were proposed demands that the situation be reanalyzed and that his hypotheses be recast. Such is the purpose of the discussion which we are now summarizing.

Hering supposes that both of the balancing vital processes of anabolism and katabolism can be augmented directly by the action of a stimulus, and he believes that he finds an explanation of sensory response, adaptation, recovery, and especially of antagonism in the quantitative consequences of this supposition. These consequences constitute a theory of sensory equilibria, and according to Hering's doctrine sensation appears only in conjunction with changes in the position of these equilibria, shifts in opposite directions producing antagonistic sensations. However, the assumptions of this theory are inconsistent with the modern belief that the immediate process of stimulation is always katabolic, and many details in the consequences of the theory are at variance with fact.

Accordingly, an attempt is made to redevelop the metabolism of response along more modern lines. The postulates and the deductions from them are presented in mathematical form, and lead to a number of characteristic equations which are descriptive of the conditions and processes of simple chemical response. These equations are based upon the supposition that stimulation can directly influence katabolism only, and that the intensity of sensation depends upon that of the katabolic change in the sense-organ. However, a certain amount of katabolism is supposed to occur in the absence of a stimulus and this constitutes the " self-excitation " of the sensory system. On the other hand, the inherent repair process which necessarily exists as the ground of the katabolism may also be appreciably increased in the presence of a strong stimulus. When such increase occurs the response is described as " compensating."

The equations of response thus obtained are interpreted, and are then applied to the experimental facts of sensory psychology. Among these facts the most significant is asserted to be *adaptation*. As a result of our theoretical discussion we have been led to the view that adaptation may be used as a criterion of the presence of chemical mechanisms underlying sensation. The several senses are examined in some detail, with the outcome that those of touch, temperature,[40] motor activity, taste, and vision are found to be metabolic. The author expresses the opinion that the mechanism of olfaction is one peculiar to itself, although chemical, as commonly supposed. Audition and the labyrinthine senses appear to be purely mechanical and non-adaptive forms of response.

In the study of the senses of temperature, taste, and vision we find ourselves forced to explain the phenomenon of an-

tagonism between certain qualitative different stimuli. This is accomplished in the present discussion by means of a mathematical assumption which differs from that of Hering, but which leads to the same general results.

It is suggested furthermore that the affective processes have a metabolic ground in the activities of the brain, and that affective adaptation is evidence for this view.

Hering's theory, then, although mistaken in the particular form of its postulates, and hence unfortunate in its attempted applications, contains a great deal of general truth, and should receive the correction and special elaboration which it has been the purpose of the present paper only to begin.

APPENDIX

1. This theory is given in its most general form in:
   HERING, E. Zur Theorie der Vorgänge in der lebendigen Substanz. *Lotos,* IX., 1888.
2. See:
   HERING, E. *Zur Lehre vom Lichtsinne,* Wien, 1878, pp. 74-85.
3. See, for example:
   FICK, A. Kritik der Hering'schen Theorie der Lichtemfindung. *Sitzungsbericht der phys.-med. Gesellschaft.* Würzburg, 1900.
4. The equation given is a "mass action" equation. Like (3) it depends upon the fact that the chances that a certain number of molecules of the sensitive substance will be decomposed in a given time are proportional to the total number present.
5. See reference 1, above: *"Sonderabdruck,"* p. 2.
6. See reference 1, above: *"Sonderabdruck,"* p. 4.
7. It is obvious that equation (7) specifies the conditions of an equilibrium between the cell and the lymph with respect to a transfer of the substance, *S*. Such an equilibrium exists when the cell is emitting as many molecules of *S* per second as it receives. However, this condition does not necessarily imply that the concentration in the cell is the same as that in the lymph at this time. As a matter of fact, even in the unstimulated cell this equilibrium will never be realized on account of the autonomous katabolism which is going on within the cell.
8. The equation of adaptation is derived as follows: Take $(c_k + c_d i) = b$. Then, by (4) and the definition of $\dot{s}$,
   $$ds/dt = a - bs \quad \text{and} \quad -ds/dt = bs - a.$$
   Integrating the last expression; we get
   $$-\int \frac{ds}{bs - a} = \int dt + c \qquad \text{or}$$
   $$-\frac{1}{b} \log (bs - a) = t + c$$

But $c = o$, so that, transposing;

$log\ (bs - a) = -bt$, or, in exponential form:

$$bs - a = e^{-bt} = \frac{1}{e^{bt}}, \text{ whence:}$$

$$s = \frac{1}{b}\left[a + \frac{1}{e^{bt}}.\right]$$

On substituting this value of s in (6), we get:

$$q = a + \frac{1}{e^{bt}},$$

the equation of adaptation.

9. The equation of recovery was derived in the following manner: From equations (a) and (c) we have, when $i = o$:

$$ds/dt = a - c_k s$$

Transforming and integrating, we get:

$$\int \frac{ds}{a - c_k s} = \int dt + c \qquad \text{or}$$

$$-\frac{1}{c_k}\ log\ (a - c_k s) = t + c$$

Putting this into exponential form, we have if $c = o$:

$$a - c_k s = e^{-c_k t} \qquad \text{or}$$

$$s = \frac{1}{c_k}\left[a - \frac{1}{e^{c_k t}}\right]$$

Substituting this in the equation, $q = c_k s$, we obtain:

$$q = a - \frac{1}{e^{c_k t}}$$

It is obvious, however, that the form of this function must depend to a certain extent upon the value of s for which t is taken as zero. If we place $t = o$ in the equation as written we have: $q = a - 1$, from which:

$$s = \frac{a - 1}{c_k}$$

so that for the equation in the form given the recovery time must be measured from the instant at which s has the above value.

10. *vide infra.*
11. The writer desires to state explicitly that the present paper is regarded by him merely as a preliminary attempt towards an application of the method of mathematical hypothesis to the problems of sensory response. It is his opinion that this method would prove exceedingly useful in psycho-physics, if its nature and significance were clearly understood. Mathematical reasoning need not be limited to subject-matter which is capable of exact measurement. Its most general utility lies in the development and expression of *types of interdependence* between variable quantities. So long as we can *recognize the type* in the phenomena to which the mathematical results are applied, these results have significance. The reasoning presented in the present paper is in every sense schematic, and the hypotheses are tentative, although they are employed with that degree of confidence which is essential in any courageous attempt to discover the truth.
12. *Cf.*, for example:
    SCHÄFER, K. L. Der Gehörsinn, in Nagel's *Handbuch der Physiologie des Menschen,* III, 1905, p. 512.
13. See reference 1, "*Sonderabdruck,*" pp. 18 ff.
14. See, for example:
    LILLIE, R. S. The Relation of Stimulation and Conduction in Irritable Tissues to Changes in the Permeability of the Limiting Membranes. *American Journal of Physiology,* XXVIII, 1911, pp. 197-223.
15. For a definite denial of auditory fatigue, and a rather detailed discussion of the experimental evidence upon this point, see Schäfer, *loc. cit.,* pp. 506-512. A series of experiments to prove auditory fatigue, but with negative results, is described by:
    SEWALL, E. Zur Lehre von der Ermüdung des Gehörorgans. *Zeitschrift für Sinnesphysiologie,* XLII, 1907-1908, pp. 115-123.
    This investigator's method is the same as that of Urbantschitsch (1881) and Thompson (1881) who believed they had proven the existence of such fatigue.
16. MEYER, M. Theorie der Gerauschempfindungen. *Zeitschrift für Psychologie und Physiologie der Sinnesorgane,* XXXI, 1903, pp. 233-247.
17. See:
    TITCHENER, E. B. *A Text-book of Psychology,* 1910, p. 154.
18. See reference 17, p. 157.
19. There is some dispute as to whether touch adaptation is due to peripheral or to central causes, since it increases in the case of certain lesions in the cortex. However, it is a fairly safe policy to regard a process as peripheral until it has been proven to be central. On this, and related questions, see:
    THUNBERG, T. Physiologie der Druck-, Temperatur-, und Schmerzempfindungen; in Nagel's *Handbuch der Physiologie des Menschen,* III, 1905, pp. 647-735.
20. FECHNER, G. T. *Elemente der Psychophysik,* 2te unveränderte Auflage, II, 1889, pp. 311 ff.
21. See Thunberg, *loc cit.,* pp. 668-669.
22. HERING, E. Der Temperatursinn; in Hermann's *Handbuch der Physiologie,* III, 1879, (2), pp. 415-440.

23. It should be noticed that here the usual criticism of the Hering theory is not supported by chemical analogy. In general, decrease in temperature certainly does favor chemical synthesis. However, there is no evidence that effects of this sort in the living organism can directly produce sensation.
24. HEAD, H., and RIVERS, W. H. R. A Human Experiment in Nerve Division. *Brain,* XXXI, 1908, pp. 323-451, esp. Chap. VII.
25. *Loc. cit.,* pp. 442-446.
26. BABÁK, E. Ueber die Temperaturempfindlichkeit der Amphibien. *Zeitschrift für Sinnesphysiologie,* XL, 1912, pp. 34-45, believes that he has evidence of a neural polarity of the heat and cold systems, in the nature of the reactions which heat and cold produce in the frog.
27. VOIGT, A. Ueber die Beurteilung von Temperaturen unter dem Einfluss der Adaptation. *Zeitschrift für Psychologie und Physiologie der Sinnesorgane,* LVI, 1910, pp. 344-378, denies that temperature adaptation is complete, but it is to be doubted if the periods of adaptation which he employed in his experiments were adequate to prove his thesis.

    RUBIN, E. Beobachtungen über Temperaturempfindungen. *Zeitschrift für Sinnesphysiologie,* XL, 1912, pp. 388-393, finds that temperature sensations exist only so long as the temperature of the skin is undergoing change. He proved by thermometric methods that such change may continue under constant conditions, for as long as an hour.
28. The investigations of Rubin, quoted in the preceding note, proved that the intensity of temperature sensation is greater when the rate of change of the temperature of the skin is greater. This is in harmony, of course, with Hering's theory, and it can be shown mathematically that it is also consonant with the hypotheses of the present paper, can in fact be deduced from them.
29. Some doubt must be felt as to whether the limits given are actually applicable to the temperature sensations themselves, since the extremes of temperature pass over psychologically into pain.
30. NAGEL, W. *Handbuch der Physiologie des Menschen,* III, 1905, p. 613.
31. See Nagel, *op. cit.,* p. 643.
32. On these points, see Nagel, *op. cit.,* pp. 641-644.
33. Simultaneous as well as successive contrast occurs in the case of taste, but it is not our purpose to consider the basis of such contrast in the present paper, although it finds ready explanation in terms of the metabolic theory of sensory response. See the author's paper in the *American Journal of Physiology,* XXXII, 1913, pp. 34-36, for a statement of this explanation in the case of vision.
34. Hering's visual hypotheses are well stated in his *Zur Lehre vom Lichtsinne,* reference 2, above. For his discussion of visual fatigue and recovery, see:

    HERING, E. Ueber Ermüdung und Erholung des Sehorgans, Leipzig, 1891.

35. MÜLLER, G. E. Zur Psychophysik der Gesichtsempfindungen. *Zeitschrift für Psychologie und Physiologie der Sinnesorgane,* XIV, 1897, pp. 1 and 161.
36. TROLAND, L. T. A Definite Physico-chemical Hypothesis to Explain Visual Response. *American Journal of Physiology,* XXXII, 1913, pp. 8-41.
37. For experimental data showing the nature of the curves, see:
    KRIES, J. VON. Die Gesichtsempfindungen; in Nagel's *Handbuch der Physiologie des Menschen,* III, 1905, p. 216.
    The curve of adaptation which is given has the right form if the relationship between the intensity of the stimulating light and the times of stimulation is taken into consideration. The curve of recovery is less satisfactory, but its lack of continuity throws doubt upon its validity.
38. In addition to the reference given in note 37, see, on adaptation in the cones:
    NAGEL, W. Helmholtz's *Physiologiche Optik,* 3te Auflage, II, pp. 282-283.
    On adaptation in the rods (visual purple) see the same, pp. 264-278. Also:
    PIPER, H. Über Dunkeladaptation. *Zeitschrift für Psychologie und Physiologie der Sinnesorgane,* XXXI, 1903, pp. 161-215.
39. On affective adaptation see:
    TITCHENER, E. B. A Text-book of Psychology, 1910, pp. 229-230, and Lectures on the Elementary Psychology of Feeling and Attention, 1908, pp. 65-69.
40. Evidence that the cutaneous senses are chemical has been found by:
    MOORE, T. V. The Influence of Temperature and the Electric Current on the Sensibility of the Skin. *Psychological Review,* XVII, 1910, pp. 347-380.